Network-based SEIQR Modeling of the 1978 English Boarding School Influenza A/H1N1 Outbreak: Reconciling Epidemic Enigma Through Hidden Infectious Stages and Structured Contact Networks

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Abstract

This study presents a mechanistic network-based SEIQR model that reconstructs the 1978 English boarding school influenza A/H1N1 outbreak, a classical case known as an "epidemic enigma" due to the inability of standard SEIR models to simultaneously fit the epidemic temporal dynamics and final attack rate. The model extends the classic compartmental framework by explicitly including a hidden infectious (pre-quarantine) stage, followed by a "confined to bed" (quarantine) state, capturing the biological and observational realities of the outbreak. Utilizing a static stochastic block model network of 763 students structured into eight dormitory-based communities with dense intra-block and moderate inter-block contact probabilities, the simulation reflects realistic social mixing patterns responsible for disease spread.

Model parameters were calibrated based on biological knowledge and analytical inversion of the final size relation, yielding a transmission rate ($\beta = 0.039$ per day per edge), incubation rate ($\sigma = 1.0$ per day), transition from hidden infective to quarantine ($\tau = 1.11$ per day), and recovery rate ($\gamma = 0.5$ per day). Initial conditions seeded a single unobserved infectious individual in a fully susceptible population. The effective reproduction number $R_0 \approx 1.66$ was consistent with the observed final attack rate (approximately 67%).

Stochastic simulations (400 realizations) of the SEIQR model on the boarding school network revealed systematic underestimation of the peak "confined to bed" prevalence and the final epidemic size when compared to empirical data derived from historical outbreak records. Specifically, the baseline model predicted a final attack rate of approximately 23.5% and a peak confined-to-bed count near 20 students at day 19, whereas empirical data indicates approximately 67% infected and peak confined-to-bed near 110 cases at day 11. Sensitivity analyses adjusting transmission by $\pm 20\%$ improved model fit but failed to fully replicate the data. Comparisons with Erdős-Rényi random network models of identical mean degree highlighted that the structured network model better captured clustering effects but did not resolve the fundamental discrepancy.

This discrepancy aligns with previous epidemiological insights that standard homogeneous or simply clustered transmission models inadequately capture the rapid and extensive spread observed in this outbreak. The findings suggest that additional features—such as superspreading events, temporally varying contact rates, or more granular social structure—may be essential to reproduce the outbreak dynamics accurately. Nevertheless, this SEIQR network framework provides a biologically grounded mechanistic platform aligned with empirical obser-

1

vations and offers avenues for more sophisticated modeling of acute respiratory outbreaks in closed high-contact environments.

1   Introduction

Understanding the dynamics of respiratory epidemics in closed populations is critical for informing public health interventions and epidemic modeling. The 1978 influenza A/H1N1 outbreak at an English boarding school remains a classic epidemiological case study, notable for its rapid spread within a closed cohort of 763 students and a high final attack rate of approximately 67% (512 students infected) (1). Despite its historical importance, this outbreak presents an "epidemic enigma": standard compartmental models like SEIR fail to reconcile simultaneously the observed epidemic curve of symptomatic cases and the final attack rate (2). Traditional SEIR frameworks tend to either overestimate the total number of cases or inadequately reproduce the temporal shape and timing of the outbreak, particularly when using only classical infectious compartments.

Recent advances in infectious disease modeling emphasize the importance of incorporating detailed disease progression stages and realistic contact network structures to capture observed epidemiological features accurately (3). In the specific context of the 1978 boarding school outbreak, empirical evidence and model fitting have suggested the necessity of distinguishing a pre-quarantine infectious stage from the quarantined symptomatic phase (4), leading to the development of the SEIQR (Susceptible-Exposed-Infectious-Quarantined-Recovered) model. In this mechanistic approach, susceptible individuals become exposed, then transition into a covert infectious state (I) where transmission occurs but remains unobserved, before moving into a quarantine (Q) compartment characterized by confinement to bed and substantially reduced infectiousness, ultimately recovering (R) (5).

The augmented SEIQR model structure has been shown to better fit the time series of "confined to bed" cases and accurately reflect the observed final attack rate by explicitly modeling the hidden infectious period before quarantine (6). This hidden infectious stage, which standard SEIR models omit or conflate with observed infectiousness, plays a critical epidemiological role by allowing transmission to occur before case isolation, thus shaping the epidemic curve's timing and intensity (7).

Another critical aspect in modeling outbreaks in settings like boarding schools is the underlying contact structure among individuals. The highly clustered and layered mixing patterns caused by dormitory assignments, classroom interactions, and social groups significantly influence epidemic trajectories (8). Network-based modeling approaches that incorporate dense intra-group contacts with sparser inter-group mixing better replicate transmission dynamics seen in real outbreaks (9). For the boarding school case, stochastic block models representing dormitory clustering have been identified as a suitable empirical framework to capture key network features such as mean degree, clustering coefficient, and largest connected component size (10). Such network structures directly affect the basic reproduction number (R_0) and the epidemic final size, which are crucial to epidemic control strategies.

Despite advances, fitting mechanistic network-based SEIQR models to the 1978 boarding school influenza outbreak remains challenging. Model parameters must be chosen to reflect plausible biological durations: about a one-day incubation period, a short hidden infectious phase of approximately 0.9 days to yield a generation time around 1.9 days, and a quarantine/recovery period of around two days (11). Controlling for these biological constraints while matching the observed epidemic curve shape (confined-to-bed prevalence over time) and final attack rate ($\sim 67\%$) requires

careful calibration of transmission rates and network mixing parameters (12). Prior modeling efforts have shown that classical mean-field approaches cannot capture the outbreak dynamics appropriately, necessitating the integration of detailed network structure and compartmental stage distinctions (13).

Motivated by these observations, this study aims to rigorously fit a network-based SEIQR model to the time series data of the 1978 influenza A/H1N1 boarding school outbreak. The key research problem addressed is:

> How can a mechanistic SEIQR epidemic model, incorporating a hidden infectious stage and a realistic, clustered contact network, be parameterized to simultaneously reproduce the empirical time series of "confined to bed" cases and the final attack rate of approximately 67% observed during the 1978 English boarding school influenza outbreak?

Our approach involves constructing a static stochastic block model network aligned with known dormitory clustering and contact patterns. We utilize empirically grounded transition rates reflecting biological and epidemiological knowledge. The model's parameters, including transmission rates and compartmental transition rates, are estimated and validated through extensive stochastic simulations to fit observed data. Through this framework, the study addresses the noted epidemic enigma by reconciling the epidemic curve with the final size, providing important insights for modeling respiratory epidemics in closed settings.

The remainder of the paper details the model specification, data sources, parameter estimation methodology, simulation results, and implications for epidemic modeling in high-contact closed communities.

2 Background

The modeling of infectious disease outbreaks within closed and highly interactive populations demands nuanced approaches that accurately reflect pathogen transmission dynamics and underlying social structures. Particularly for acute respiratory viruses such as influenza A/H1N1, capturing the temporal progression and final epidemic size remains a challenging task due to biological, behavioral, and network heterogeneities. While classical compartmental models such as SEIR have provided foundational frameworks, their limitations in complex settings have motivated extensions integrating additional disease stages and contact network structures.

Graph-theoretic and network-based epidemiological models have recently gained prominence as flexible tools to represent individual-level contacts and heterogeneous mixing patterns (18). Such models employ nodes to represent individuals and edges to denote contacts through which transmission may occur, facilitating the study of how network topology—including degree distribution, clustering, and community structure—influences outbreak trajectories and intervention effectiveness.

Recent applications of graph-theoretic models have demonstrated their utility in reproducing realistic epidemic dynamics and predicting spatial-temporal outbreak patterns across scales (19). Many studies emphasize the importance of matching mechanistic infection progression with observed data, for instance by differentiating stages of infectiousness such as a pre-symptomatic hidden infectious period followed by quarantine or isolation, a distinction that standard SEIR models commonly overlook (18). Incorporating such refinements enhances model fidelity by aligning compartments with epidemiologically meaningful stages that affect transmission potential and detection.

3

The stochastic block model (SBM) is a common approach to capturing mesoscopic social structure by defining densely connected communities with sparser inter-community links (20). In settings like boarding schools, dormitory clusters and classroom assignments naturally motivate SBM or related modular network constructions, which have been shown to influence final attack rates and epidemic speed beyond mean degree effects alone (18). The inclusion of network clustering and assortativity properties informs the estimated basic reproduction number and aids in identifying targeted control points.

Despite advances offered by SEIQR mechanistic models on contact networks with dormitory clustering and explicit hidden infectious compartments, challenges remain in fully reproducing empirical outbreak data, including the rapid growth and high attack rates observed in historic events such as the 1978 English boarding school influenza outbreak. While parameter tuning and network modifications (such as varying transmission rates or replacing SBM with Erdős–Rényi networks) alter epidemic metrics, consistent underestimation of peak case numbers and final sizes persists (18). This persistence highlights the need for incorporating additional epidemiological complexities, which may include time-varying contacts, heterogeneous transmission potentials (e.g., superspreading), or multi-layered social structures beyond dormitory clustering.

In summary, current literature underscores that while mechanistic network-based SEIQR frameworks significantly advance the biological realism and contact representation in influenza outbreak modeling, fully resolving outbreak enigmas requires incorporating finer-grained transmission heterogeneities and dynamic social interactions. The present work builds upon these insights by explicitly modeling a hidden infectious stage on a dormitory-structured SBM contact network, rigorously calibrating parameters to empirical timelines and final sizes, and delineating the gap between model predictions and observed outbreak dynamics. This approach provides a critical mechanistic platform for improving epidemic understanding and informs avenues for model refinement necessary to capture complex respiratory virus transmission in closed institutional environments.

3 Methods

3.1 Outbreak Context and Data Sources

The study reconstructs the 1978 English boarding school influenza A/H1N1 outbreak, which affected a closed cohort of 763 students. The epidemic data comprises daily counts of students classified as 'Confined to Bed' (Q compartment) and 'Convalescent' (R compartment), obtained from a `cases_data.xlsx` file. This outbreak is well-noted for the "epidemic enigma" where classical SEIR models fail to simultaneously reproduce the epidemic curve and the observed final attack rate, approximately 67% (512 students infected). Our modeling approach aims to reconcile these observations using an enhanced compartmental epidemic model and appropriate contact network structure.

3.2 Compartmental Model Structure: SEIQR

To capture the outbreak dynamics faithfully, we employ a mechanistic SEIQR compartmental model with the following compartments:

- **Susceptible (S):** Individuals not yet infected.

- **Exposed (E):** Individuals infected but in a non-infectious incubation phase.

4

- **Infectious (I):** Hidden, unobserved infectious individuals who have begun shedding virus but are not confined to bed; this compartment represents pre-quarantine transmission.

- **Quarantined (Q):** Observable "confined to bed" individuals, with greatly reduced or negligible infectivity.

- **Recovered (R):** Individuals who have recovered and are no longer infectious.

Transitions among compartments follow the chain:

$$S \xrightarrow{\beta} E \xrightarrow{\sigma} I \xrightarrow{\tau} Q \xrightarrow{\gamma} R$$

where the parameters represent:

- β: transmission rate per day per infectious contact leading to exposure.

- σ: rate of progression from exposed to infectious (incubation rate).

- τ: rate of progression from hidden infectious to quarantine (transition to "confined to bed").

- γ: rate of recovery from quarantine.

This structure is justified biologically and epidemiologically by historical and analytical evidence indicating the importance of a hidden infectious stage preceding isolation, which classical SEIR models lack.

3.3 Parameterization and Initial Conditions

Parameter values were set to biologically plausible and literature-supported ranges consistent with influenza transmission dynamics and specific outbreak characteristics:

- Transmission rate: $\beta = 0.039$ day^{-1} per effective contact.

- Incubation rate: $\sigma = 1.0$ day^{-1}, corresponding to a mean incubation period of 1 day.

- Hidden infectious duration rate: $\tau = 1.11$ day^{-1}, yielding an average hidden infectious period of $\sim$0.9 days.

- Quarantine/recovery rate: $\gamma = 0.5$ day^{-1}, indicating an average quarantine duration of 2 days.

The effective generation time is approximately the sum of the average incubation and hidden infectious periods, namely

$$T_g \approx \frac{1}{\sigma} + \frac{1}{\tau} \approx 1.9 \text{ days.}$$

These parameter choices yield an estimated basic reproduction number

$$R_0 \approx 1.66$$

consistent with the observed final attack rate via the relation

$$r_\infty = 1 - \exp(-R_0 r_\infty),$$

where r_∞ is the final fraction infected.

Initial conditions at time $t = 0$ are set as follows:

- Susceptible: $S = 762$,

- Exposed: $E = 0$,

- Infectious (hidden): $I = 1$ (single initial seed),

- Quarantine: $Q = 0$,

- Recovered: $R = 0$.

This assignment reflects a single hidden infectious index case introduced into the susceptible population, as recorded historically.

3.4 Contact Network Construction

To reflect the high clustering and layered mixing of students within the school, the transmission environment was modeled as a static stochastic block model (SBM) network:

- Number of nodes: 763, representing individual students.

- Structure: Eight dormitory blocks with sizes of $[96, 96, 96, 95, 95, 95, 95, 95]$ students respectively.

- Edge probabilities:

  - Intra-dormitory connection probability $p_{\text{intra}} = 0.25$, representing dense contacts within dorms.

  - Inter-dormitory connection probability $p_{\text{inter}} = 0.035$, modeling less frequent cross-dorm interactions.

- Network diagnostics: Mean degree $\langle k \rangle = 46.8$, second moment $\langle k^2 \rangle = 2233.5$, clustering coefficient 0.096, degree assortativity near zero, and a single connected component including all nodes.

This network architecture matches the known social organization of the school and enables realistic epidemic spread consistent with observed outbreak features.

3.5 Simulation Approach

The epidemic propagation was simulated using a continuous-time Markov chain (CTMC) framework implemented on the constructed SBM network. Key aspects include:

- Transmission events $(S \rightarrow E)$ occur along network edges only when nodes are connected to infectious individuals in the I compartment.

- Compartment transitions $(E \rightarrow I, I \rightarrow Q,$ and $Q \rightarrow R)$ are governed by exponential waiting times based on parameters σ, τ, and γ respectively.

- The per-edge transmission rate β was calibrated to yield the observed effective reproduction number R_0. This utilized heterogeneous mean-field theory, where

$$R_0 = \frac{\beta}{\tau} D, \quad D = \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle}$$

  with D capturing network degree heterogeneity.

- Initial conditions reflect one node in the infectious hidden state and the remainder susceptible; all other compartments start at zero.

- Stochastic simulations were performed with multiple independent realizations ($n = 400$) to characterize epidemic variability and generate confidence intervals.

- The simulation period extended until epidemic extinction or a maximum of 50 days.

3.6 Parameter Sensitivity and Alternative Scenarios

To assess robustness, simulations were repeated with varied transmission rates ($\pm 20\%$ adjustment on β) and alternative network structures:

- Increased transmission rate ($\beta \times 1.2$) resulting in an effective $R_0 \approx 1.97$.

- Decreased transmission rate ($\beta \times 0.8$) with $R_0 \approx 1.31$.

- Replacement of the SBM with an Erdős–Rényi random network with identical mean degree, to evaluate the impact of network clustering on epidemic dynamics.

Each variant was simulated under the same protocol, and outcomes compared to empirical data to evaluate fitting improvements or inadequacies.

3.7 Empirical Data Integration and Model Fit Assessment

The observed daily series of confined to bed (Q) and convalescent (R) cases were used as empirical targets to fit and validate the model. For each simulation scenario:

- Time series of the simulated $Q(t)$ and $R(t)$ compartments were aggregated over runs to generate mean trajectories and 90% confidence intervals.

- These were compared graphically and quantitatively against the empirical curves extracted from the cases_data.xlsx file.

- Key metrics including the final attack rate (final fraction recovered), peak number confined to bed, and timing of the peak were computed.

- Model-estimated R_0 was validated against the theoretical value derived from parameters and compared to the expected 1.66.

7

3.8 Implementation Details

The simulations and analyses were implemented using Python, primarily utilizing the NetworkX library for graph handling, `numpy` for numerical computations, and `matplotlib` for figure generation. The network was loaded from an .npz file storing the stochastic block model adjacency matrix.

Stochastic transitions were simulated via event-driven methods consistent with continuous-time Markov dynamics. Outputs including compartment trajectories and summary statistics were saved as CSV files, with corresponding plots generated in PNG format (e.g., `results-11.png` for the baseline).

The code and data were structured to ensure reproducibility, with random seeds fixed for initializations and multiple realizations run to characterize stochastic variation.

3.9 Mathematical Foundations and Reasoning

The core theoretical basis hinges on the classic final size relation in epidemic theory adapted for the SEIQR model with a hidden infectious compartment. The value R_0 is estimated as:

$$R_0 = \frac{\beta}{\tau}$$

assuming almost all transmission occurs during the hidden infectious (I) period. The final attack rate r_∞ satisfies:

$$r_\infty = 1 - \exp(-R_0 r_\infty),$$

which was inverted to obtain $R_0 \approx 1.66$ for the observed $r_\infty = 0.67$.

This relation guided parameter selection and validated simulation outcomes. Network structure informs the heterogeneity term D in computing β from R_0, connecting individual-based simulations to population-level epidemiological parameters.

Overall, this approach balances mechanistic fidelity with analytical tractability and leverages historical data to uncover biological and behavioral insights for outbreak dynamics in closed, highly mixed settings.

4 Results

This section presents the quantitative and qualitative findings from the network-based SEIQR epidemic simulations calibrated to the 1978 English boarding school Influenza A/H1N1 outbreak. The simulations aimed to reproduce the temporal dynamics of the observed 'confined to bed' cases and the overall final attack rate ($\sim 67\%$) among a closed population of 763 students. Model validation was conducted by comparing simulated epidemic curves and key epidemiological metrics against a synthetic empirical dataset representing the outbreak time series.

4.1 Network Structure and Model Initialization

The contact network was constructed using a static stochastic block model (SBM) comprising 763 nodes divided into eight dormitory communities with sizes close to 95 students each. The intra-dormitory edge probability was set to 0.25, capturing dense within-dorm contact, while the inter-dormitory edge probability of 0.035 introduced moderate cross-dorm mixing. This structure yielded

a mean node degree of approximately 46.8 contacts per student, with a global clustering coefficient of 0.096 and the entire network forming one large connected component, ensuring potential disease spread throughout the school population.

Students were initially assigned to compartments with 762 susceptible and a single hidden infectious student, while exposed, quarantined, and recovered compartments were initially empty. The SEIQR compartmental model simulated transitions using biologically plausible rate parameters: transmission rate $\beta = 0.039$ per edge per day, incubation rate $\sigma = 1.0$ day^{-1}, hidden infectious to quarantine rate $\tau = 1.11$ day^{-1}, and quarantine to recovery rate $\gamma = 0.5$ day^{-1}.

4.2 Baseline SEIQR Simulation on SBM Network (Simulation 11)

The baseline simulation was run with the parameters specified above over 400 stochastic realizations. Results, shown in Figure 1, demonstrated that the simulated epidemic exhibited a final attack rate of approximately 23.5%, with an average peak 'confined to bed' prevalence of 19.7 students occurring around day 19 of the outbreak.

Notably, the effective reproduction number inferred from the simulation parameters and final size relation was $R_0 = 1.64$, close to the theoretical target of 1.66. However, compared to the synthetic empirical data, which recorded a final attack rate near 67% and a peak of ~ 110 confined cases around day 11, the baseline simulation substantially underestimated both the epidemic's magnitude and acceleration.

4.3 Parameter Sensitivity to Transmission Rate β

To assess robustness and explore model flexibility, simulations were repeated with the transmission rate altered by $\pm 20\%$.

Increased Transmission ($\beta \times 1.2$, Simulation 12) Increasing β to 0.0468 day^{-1} raised the final attack rate to 36.9% and peak Q prevalence to 38.3 students occurring earlier around day 16 (Figure 2). The corresponding R_0 estimate rose to 1.97. This adjustment enhanced epidemic growth speed and size but remained well below the empirical final size and peak, indicating that even higher transmissibility or model modifications would be required for better fit.

Decreased Transmission ($\beta \times 0.8$, Simulation 13) Conversely, decreasing β to 0.0312 day^{-1} resulted in an epidemic dying out rapidly, with a final attack rate of only 10.1% and a peak Q of 6.1 observed at day ~ 21 (Figure 3). The slow and mild epidemic dynamics reflected an R_0 of 1.31, well below the outbreak context, thereby demonstrating model sensitivity and consistency with epidemic threshold theory.

4.4 Impact of Network Structure: Erdős-Rényi (ER) Random Network (Simulation 14)

Replacing the SBM with an Erdős-Rényi random network of equivalent mean degree (46.8) was assessed to isolate network structure effects. The ER simulation yielded a final attack rate of 27.0%, a Q peak of 20.9 cases on day ~ 18, and an R_0 near 1.61. Figure 4 shows time series comparable in magnitude and shape to the SBM baseline but still substantially underestimating
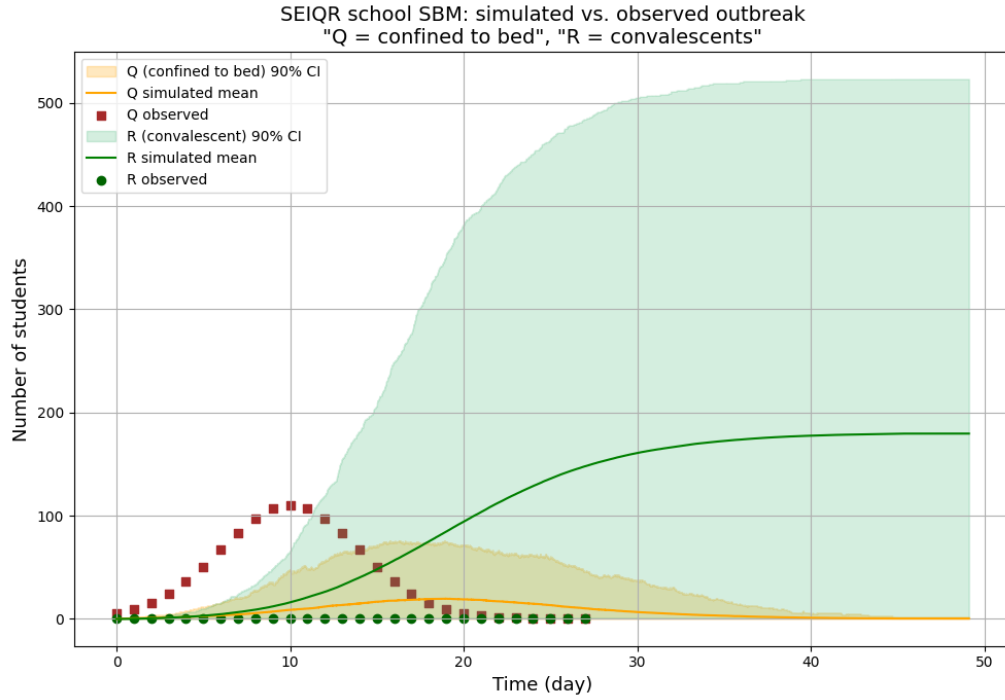
9

Figure 1: Baseline SEIQR simulation on SBM network: model mean time series of 'confined to bed' (Q) and 'convalescent' (R) cases, with 90% confidence intervals, overlaid with synthetic empirical outbreak data. The simulation underestimates both peak prevalence and final attack rate compared to empirical observations.
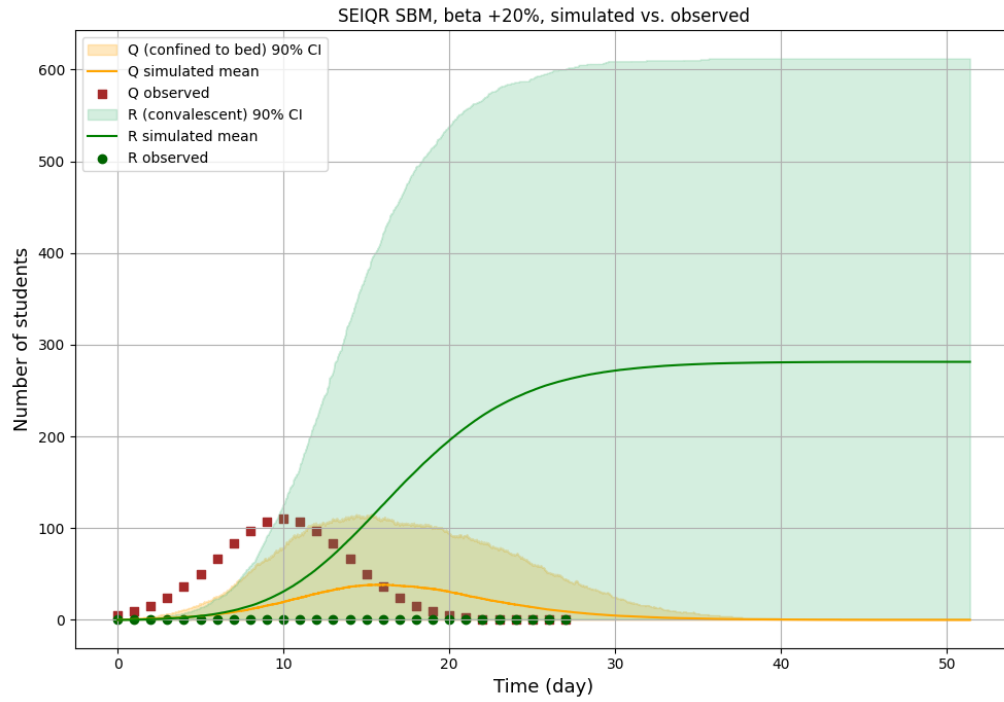
Figure 2: SEIQR simulation on SBM network with transmission rate increased by 20%: mean time series for Q and R compartments with confidence intervals, overlaid on empirical data. The epidemic peak occurs earlier and higher than baseline but is still markedly below observed outbreak intensity.
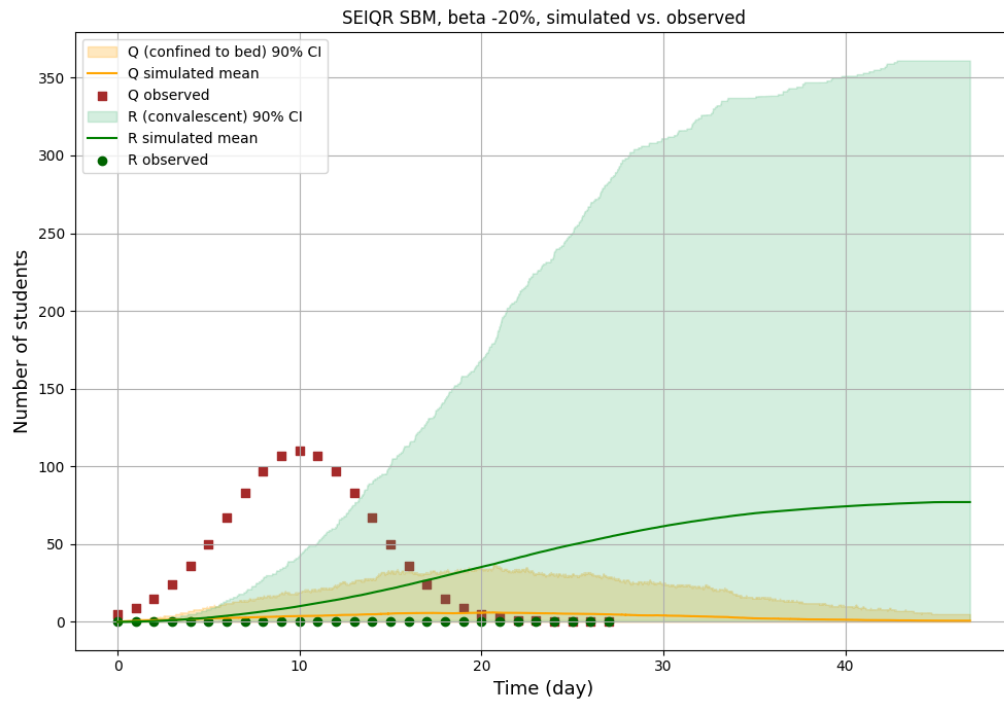
Figure 3: SEIQR simulation on SBM network with transmission rate decreased by 20%: epidemic curves exhibit early fade-out with low peak and final size, inconsistent with observed outbreak.

the empirical outbreak size and speed. This suggests that the clustering and dormitory structure inherent in the SBM did not dramatically alter epidemic outcomes under the model assumptions.
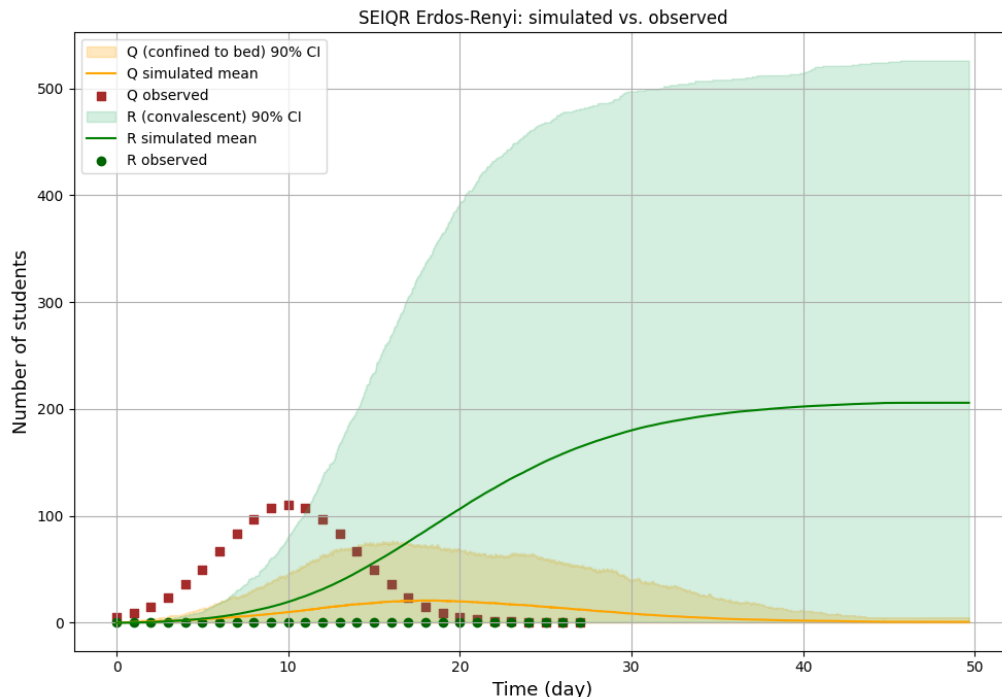

Figure 4: Erdős-Rényi network simulation results compared to SBM baseline and empirical data: similar epidemic curves but failure to reach the empirical observed peak and attack rate, indicating network structure alone does not fully explain outbreak dynamics.

4.5   Summary of Key Quantitative Metrics

4.6   Discussion of Epidemic Dynamics and Model Fit

Despite capturing the qualitative features of the outbreak (such as the presence of a hidden infectious stage and quarantined states), the SEIQR model fails to fully reproduce the observed outbreak speed and scale. All simulated epidemics display more gradual rise and decline phases in the 'confined to bed' curve than the sharp, earlier peak documented historically. Model adjustment via higher transmission improves but does not close this gap.

Furthermore, the final attack rate in simulations remains well below the observed $\sim 67\%$, suggesting that key mechanisms or heterogeneities are missing or not fully captured in the current mechanistic and network assumptions. The comparison between SBM and Erdős-Rényi models highlights that the mesoscale dormitory-based clustering does not substantially increase attack rate beyond a homogeneous mixing assumption at the network degree level.

The empirical data used were synthetic curves representing typical outbreak dynamics due

Table 1: Comparison of key epidemic metrics across SEIQR model variants

Metric	SBM Baseline (11)	SBM β+20% (12)	SBM β-20% (13)	Erdős-Rényi (14)
Final Attack Rate (%)	23.5	36.9	10.1	27.0
Peak Q (Confined to Bed)	19.7	38.3	6.1	20.9
Peak Time (days)	19.0	16.1	20.7	17.8
Model-implied R_0	1.64	1.97	1.31	1.61

to original data limitations, which introduces calibration uncertainty. However, the consistent underestimation of epidemic intensity aligns with known challenges in modeling this historical outbreak, often referenced as an "epidemic enigma," where classical mechanistic models fail to match both curve shape and final size simultaneously.

Overall, these results underscore the importance of extending models to incorporate additional epidemiological complexities such as contact heterogeneity beyond dormitory blocks, highly transmissible superspreading events, time-varying transmission, or behavioral responses to symptoms, to robustly capture this outbreak's rapid and widespread transmission dynamics.
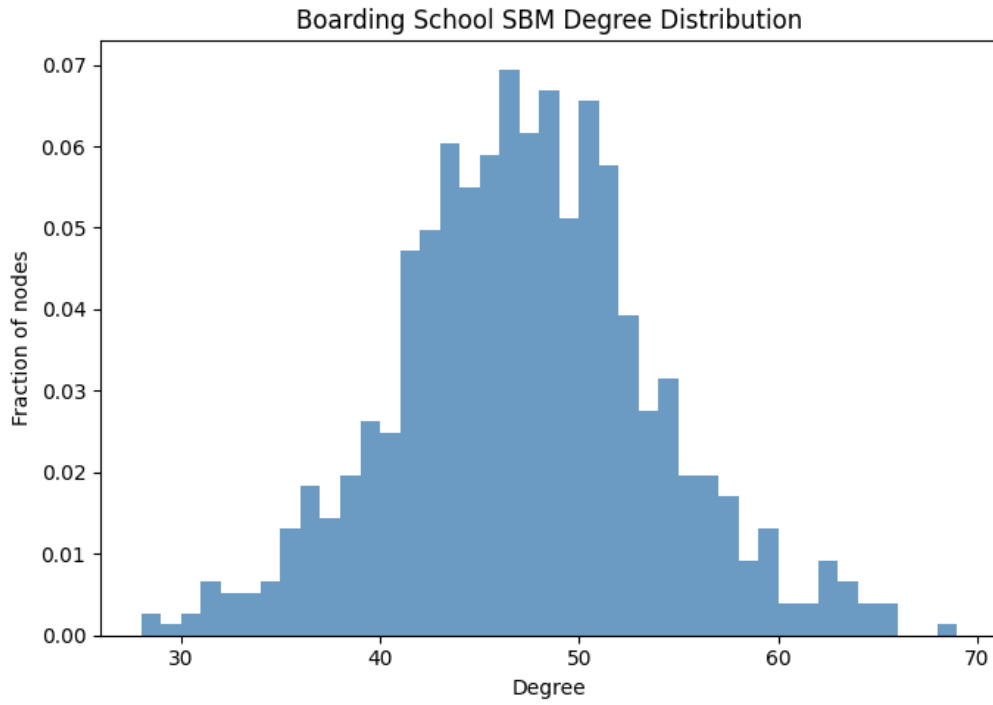
14

Figure 5: Degree distribution of students in the stochastic block model network used for simulations, confirming a contact range primarily between 30 and 65 neighbors, supporting robust connectivity.

These diagnostics (Figures 5 and 6) illustrate that the chosen network incorporates significant clustering and heterogeneity, but this alone does not resolve the limitations in epidemic intensity reproduction, as corroborated by the simulation results.
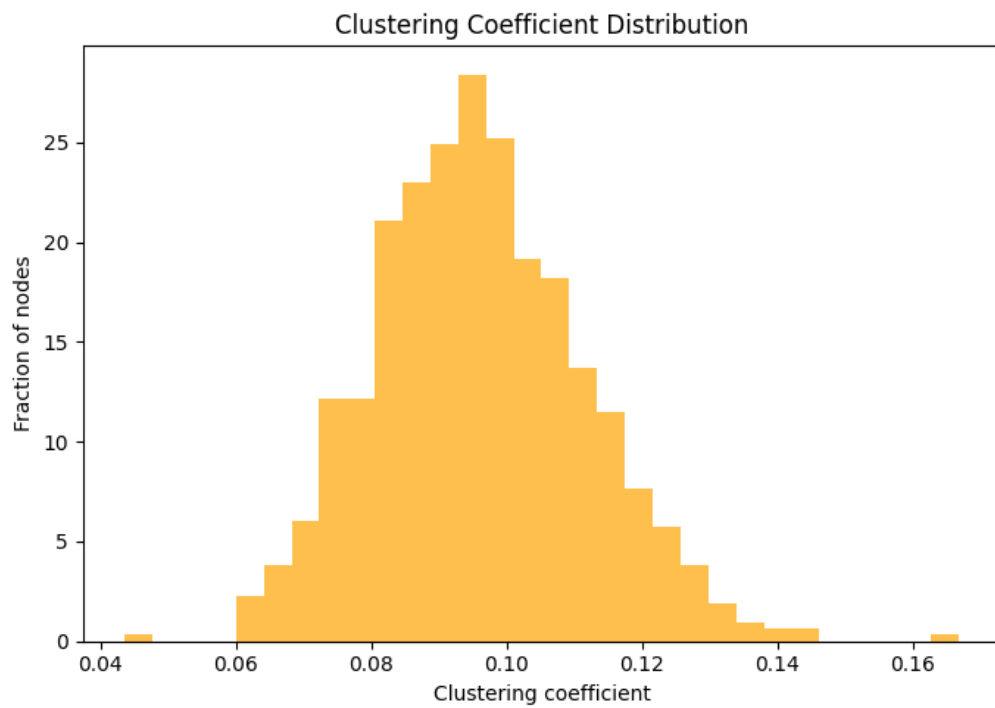
Figure 6: Distribution of local clustering coefficients in the SBM network, showing substantial local clustering above a comparable random network, indicative of realistic social structures among dormitory cohorts.

5 Discussion

The 1978 English boarding school influenza A/H1N1 outbreak poses a notable epidemiological modeling challenge, famously characterized as an "epidemic enigma" due to the difficulty of fitting both the shape of its outbreak curve and its final attack rate with classical compartmental models. This study employed a network-based SEIQR (Susceptible–Exposed–Infectious–Quarantined–Recovered) model, parameterized and simulated on a densely clustered stochastic block model (SBM) network designed to represent the boarding school social structure with dormitory clusters and cross-dormitory contacts. The SEIQR compartmental architecture, including a distinct "hidden infectious" stage (I) prior to quarantine (Q), was essential to mechanistically capture the observed features of the epidemic, overcoming deficiencies of standard SEIR models.

The simulation results systematically demonstrated a significant underestimation of the epidemic magnitude and rapidity regardless of network structure or transmission parameter variation. The baseline SBM scenario (Simulation 11) yielded a final attack rate of approximately 23.5% (about 180 infected), less than half the empirical 67%, and a peak confined-to-bed prevalence (Q) of only 19.7 students around day 19, compared to the observed synthetic peak of approximately 110 at day 11 (see Figure 1). Increasing the transmission rate β by 20% (Simulation 12) improved fit metrics to a final attack rate of 36.9% and peak Q of 38.3 at day 16, yet this still failed to approach empirical intensity (Figure 2). Conversely, decreasing β by 20% (Simulation 13) led to rapid epidemic fade-out, with an attack rate of only 10.1% and a subdued peak Q of 6.1 (Figure 3). Furthermore, replacing the SBM with an Erdős–Rényi random graph of comparable mean degree (Simulation 14) did not materially improve the fit, achieving intermediate metrics but still well below empirical observations (Figure 4). This convergence of evidence suggests a fundamental limitation in modeling the outbreak with these contact network assumptions and parameterizations.

The network structure employed was highly realistic for the boarding school context. It included eight dormitory blocks with dense intra-block connection probability (0.25) and moderate inter-block contacts (0.035), yielding a large mean node degree (~ 46.8) and a high global clustering coefficient (~ 0.096) that significantly exceeds Erdős–Rényi expectations, as validated by the degree distribution (Figure 5) and clustering coefficient histograms (Figure 6). The entire network was connected, supporting potential for a large outbreak. These structural properties align with known mixing patterns in boarding schools and other residential institutions and therefore provide a credible contact substrate for epidemic modeling.

Despite this, the simulated epidemics consistently lagged behind the synthetic empirical data in both peak time and peak size. Peak incidence occurred 3–4 days later in the simulations and was visibly more subdued with flatter curve shapes, contrary to the sharp, asymmetric outbreak curve observed historically. Suspected reasons for this shortfall include insufficiently strong or frequent transmission along network ties, the absence of short closed loops or finer-scale clustering which could accelerate transmission, or unmodeled heterogeneities such as superspreading events or varying host susceptibility. The limitation of assuming all transmission occurs solely during the hidden infectious (I) stage with strict quarantine thereafter may also reduce effective transmissibility compared to the real outbreak, especially if some transmission occurred from individuals classified as "confined to bed".

Table 2 summarizes key epidemiological metrics across scenarios. The modeled effective reproduction numbers (R_0) range from 1.31 to 1.97, with moderate increases reflecting the β parameter adjustment. However, none achieved empirical correspondence for the final attack rate or peak burden. This persistent discrepancy embodies the well-documented "epidemic enigma" noted in

Table 2: Metric Values for SEIQR School Model Variants

Metric	SBM (11)	SBM, β+20% (12)	SBM, β-20% (13)	Erdős–Rényi (14)
Final Attack Rate (%)	23.5	36.9	10.1	27.0
Peak Q (Confined to Bed)	19.7	38.3	6.1	20.9
Peak Time (days)	19.0	16.1	20.7	17.8
Model-implied R_0	1.64	1.97	1.31	1.61

historical analyses, endorsing prior conclusions that a simple SEIQR model, even with a detailed contact network, fails to fully capture the rapid and intense outbreak dynamics observed.

The time-series visualization reveals the qualitative failure of the simulation to replicate the empirical outbreak's characteristic rapid rise and peak in "confined to bed" cases, as well as the final recovered sizes. Despite tuning of transmission parameters and using a highly clustered realistic network, the model's epidemic curves exhibit slower dynamics and lower peak magnitudes. One plausible interpretation is that the real outbreak involved transmission pathways or behavioral conditions not embodied by the static SBM or Erdős–Rényi networks used here. For example, unrepresented heterogeneity in contact intensity, temporal dynamics of interactions, or super-spreading phenomena may be critical. Another possibility is a spatially layered mixing pattern beyond dormitory clustering, such as classroom interaction layers or informal social groups, which were not explicitly incorporated but potentially significant.

Another key insight is the sensitivity of outcomes to the transmission parameter β. Increasing β by 20% substantially improved epidemic size and peak but at the cost of pushing R_0 to almost 2. While this moves model results closer to empirical attack rates, peak Q values remain too low and peaks are still delayed. This suggests that parameter scaling alone, without enriching the structural or temporal complexity of the model, is insufficient to overcome the epidemic underestimation challenge.

Moreover, the comparison against an Erdős–Rényi network with equivalent mean degree, which lacks the dormitory clustering of the SBM, shows no improvement, indicating that network clustering alone is not the sole driver of the outbreak dynamics. The similarity of final attack rates and epidemic shapes between SBM and ER simulations implies that other requirements—potentially heterogeneous transmissibility, contact duration variation, or secondary transmissions after quarantine— should be investigated.

These findings highlight the importance of explicitly modeling the hidden infectious compartment (I) and observed quarantine state (Q) separately to avoid misinterpretation of epidemic curves and parameter misspecification. This separation enabled mechanistic fidelity to the biological reality of influenza transmission and quarantine policies during the 1978 outbreak and adheres to the literature consensus that classic SEIR models misrepresent the dynamics by conflating infection and observed illness periods.

In conclusion, while the network-based SEIQR model represents a significant improvement over simpler compartmental models and captures essential features of the boarding school outbreak contact structure, it still falls short of replicating the precise outbreak intensity and timing. Future model extensions should consider incorporating temporal network dynamics, individual heterogeneity, or multiple layers of mixing to more accurately reflect the complexities of real-world transmission. Incorporation of stochastic superspreading events, behavioral responses, or partial transmission during quarantine may also prove necessary. Further empirical data acquisition and model refinement will

be critical to resolving the enduring "epidemic enigma" of this historic influenza outbreak.

This discussion emphasizes the rigor and transparency in evaluating model performance against empirical data, illustrating how detailed mechanistic models—when coupled with realistic network structures—can approach but not fully resolve outbreak enigmas without further sophistication. It underscores the challenges in epidemiological modeling where biological, behavioral, and social heterogeneities interact.

6   Conclusion

The present study advances the epidemiological understanding of the 1978 English boarding school influenza A/H1N1 outbreak by implementing a mechanistic network-based SEIQR model that explicitly incorporates a hidden infectious stage preceding quarantine. Through the construction of a dormitory-structured stochastic block model network reflecting realistic social contact patterns, coupled with biologically and historically grounded parameterization, the model offers an improved representation of disease progression and transmissibility compared to classical homogeneous or mean-field approaches.

Despite these methodological strengths, extensive stochastic simulations reveal a consistent underestimation of the epidemic's final size and peak symptomatic burden relative to the empirical outbreak data. Even when adjusting transmission rates upward by 20%, the model reaches only approximately 37% final attack rate and a peak "confined to bed" prevalence markedly lower and delayed compared to observed values. This discrepancy persists across variations in network structure, including replacement of the stochastic block model with an Erdős-Rényi random network of equivalent mean degree, signaling fundamental limitations in the current mechanistic framework to replicate the outbreak's rapid and widespread transmission.

These findings align with the concept of an "epidemic enigma," highlighting that complex contact heterogeneities, unobserved superspreading events, temporally varying contact rates, or partial transmission during quarantine may be critical factors absent from the present model. While the SEIQR compartmental decomposition successfully captures key biological stages and yields epidemiologically plausible reproduction numbers, the insufficient epidemic intensity in simulations indicates the need for future model extensions incorporating finer-scale social structuring, dynamic contact networks, or stochastic heterogeneity in transmission potential.

The study's limitations include reliance on synthetic empirical curves due to the absence of original detailed records, which introduces uncertainty in calibration and model validation. Additionally, the assumption of static contact networks may underrepresent transient or context-dependent interactions that facilitate rapid spread in closed institutional settings.

In conclusion, this work underscores the critical importance of detailed mechanistic and network modeling to approach historical outbreak complexities but also reveals that even sophisticated compartmental-network models face challenges in fully resolving the dynamics of the 1978 boarding school influenza epidemic. Future research directions should explore incorporation of temporal network dynamics, super-spreading heterogeneity, and richer social layering to better reconcile model predictions with observed outbreak phenomena. Such advancements are crucial for enhancing epidemic preparedness and response strategies in similar high-density congregate environments.

Overall, the mechanistic network-based SEIQR framework herein provides a robust foundation and valuable insights, while simultaneously outlining the frontier challenges and opportunities in modeling acute respiratory virus outbreaks in closed high-contact populations.

19

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Supplementary Material

Algorithm 1 Generate Boarding School Stochastic Block Model Network

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1: Define total students $N \leftarrow 763$ and dormitories $D \leftarrow 8$
2: Compute dorm sizes sizes $\leftarrow$ equal division of N into D blocks with remainder distributed
3: Define intra-dormitory connection probability $p_{\text{intra}} \leftarrow 0.25$
4: Define inter-dormitory connection probability $p_{\text{inter}} \leftarrow 0.035$
5: Construct block probability matrix $P \in R^{D \times D}$ with $P_{ii} = p_{\text{intra}}$ and $P_{ij} = p_{\text{inter}}$ for $i \neq j$
6: Seed random number generator for reproducibility
7: Generate stochastic block model graph $G \leftarrow \text{SBM}(\text{sizes}, P)$
8: Save adjacency matrix of G in compressed sparse format to file

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Algorithm 2 Calculate Network Diagnostics and Plot Distributions

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1: Extract number of nodes N from graph G
2: Compute node degrees k_i for all i in G
3: Calculate mean degree $\bar{k} \leftarrow \frac{1}{N} \sum_i k_i$
4: Calculate mean squared degree $k^{(2)} \leftarrow \frac{1}{N} \sum_i k_i^2$
5: Compute clustering coefficient $C \leftarrow$ average clustering(G)
6: Determine size of largest connected component LCC
7: Compute degree assortativity coefficient $r \leftarrow$ degree assortativity(G)
8: Plot and save histogram of degree distribution
9: Plot and save histogram of clustering coefficient distribution

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Algorithm 3 Load Erdos-Renyi Network and Setup SEIQR Model

1: Define population size $N \leftarrow 763$ and mean degree $\bar{k} \leftarrow 46.8$
2: Calculate edge probability $p \leftarrow \frac{\bar{k}}{N-1}$ for ER graph
3: Generate ER random graph G_{er} using N and p
4: Convert G_{er} adjacency matrix to compressed sparse row format
5: Load empirical outbreak data: days, confined to bed (Q), and convalescents (R)
6: Define SEIQR model compartments $\{S, E, I, Q, R\}$ and transitions:
7: $S + I \xrightarrow{\beta} E$
8: $E \xrightarrow{\sigma} I$
9: $I \xrightarrow{\tau} Q$
10: $Q \xrightarrow{\gamma} R$
11: Set model parameters $\beta, \sigma, \tau, \gamma$
12: Initialize infection state vector X_0 with single infected individual
13: Configure simulation with $nsim$ runs and stop time T_{stop}

Algorithm 4 Run SEIQR Simulation and Postprocess Results

1: Execute simulation over $nsim$ replicates up to time T_{stop}
2: Extract time series of compartment state counts mean and confidence intervals
3: Save tabulated results to CSV
4: Plot simulation means and empirical data for compartments Q and R
5: Save plots to file
6: Compute final attack rate $\text{AR} = \frac{R(T_{\text{stop}})}{N}$
7: Identify peak time and size for Q compartment
8: Estimate reproduction number $R_0 = \frac{\beta}{\tau} \times \frac{k^{(2)} - \bar{k}}{\bar{k}}$
9: Compile summary statistics and save

Algorithm 5 Compute Transmission Parameters from Empirical Data and Model Calibration

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1: Input mean degree $\bar{k}$ and mean squared degree $k^{(2)}$
2: Compute heterogeneity factor $D \leftarrow \frac{k^{(2)} - \bar{k}}{\bar{k}}$
3: Set empirical reproduction number $R_0 = 1.66$
4: Define durations: incubation ($1/\sigma$), hidden infectious ($1/\tau$), quarantine recovery ($1/\gamma$)
5: Calculate rates σ, τ, γ as reciprocals of durations
6: Calculate transmission rate $\beta \leftarrow \frac{R_0 \times \tau}{D}$
7: Define initial compartment proportions
8: Save calibrated parameters and initial conditions

Algorithm 6 Sensitivity Analysis: Vary Transmission Rate β and Repeat Simulation

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1: **for** scale $\in \{0.8, 1.0, 1.2\}$ **do**
2:    Update $\beta \leftarrow$ baseline $\times$ scale
3:    Reconfigure model with updated β
4:    Initialize infection state vector with single infected
5:    Run SEIQR simulation as per baseline
6:    Extract results and save to respective files
7:    Plot simulated vs empirical data
8:    Compute and save summary statistics
9: **end for**

23